Bipolar catheter electrograms for study of retrograde atrial activation pattern in patients without pre-excitation syndromes

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The sequence of retrograde atrial activation of premature ventricular beats was studied in 12 patients without pre-excitation syndrome. Forward and retrograde AV nodal conduction times were within normal limits. The septal low right atrial (LRA₂) deflection was inscribed 25 to 55 ms (ave: 37.9 ms) before the low left atrial (LLA₂) electrogram and 35 to 75 ms (ave: 53.7 ms) ahead of the high right atrial (HRA₂) deflection. LLA₂ preceded HRA₂ in 8 patients, more or less coincided with HRA₂ in 3 cases, and was inscribed 10 ms ahead in 1 patient. In all cases a decrease in the coupling (St_1-St_2) interval resulted in a proportional increase of the septal LRA₁-LRA₂, LLA₁-LLA₂, and HRA₁-HRA₂ intervals. Atrial echoes occurring in 3 cases had a sequence of arrival of excitation at the recording sites which was similar to the ones shown by the same patients during ventricular pacing. This is in keeping with the existence of an upper common pathway located above the area where functional longitudinal dissociation occurred.

Thus, it is concluded that impulses emerging from the AV node in a retrograde direction reach the septal low right atrium before they activate the explored low left atrial sites, regardless as to whether they arise in the ventricles or in the atria.

There have been several reports in which the sequence of atrial activation during retrograde (VA) conduction through accessory pathways has been discussed (Latour and Puech, 1957; Torresani et al., 1969, Grolleau et al., 1970; Spurrell, Krikler, and Sowton, 1974b; Svenson et al., 1975; Gallagher et al., 1975; Tonkin et al., 1975a, b). In contrast, few studies have dealt with the usefulness of right and left atrial electrograms in analysing the pattern of atrial activation of impulses reaching the atria retrogradely through the AV node in patients without pre-excitation syndrome (Nicolai et al., 1970; Castellanos, Castillo, and Myerburg, 1971b; Svenson et al., 1974; Gallagher et al., 1975; Wu et al., 1975).

Subjects and methods

The technique used in our department for analysis of the electrical activity of the His bundle and several atrial sites has been discussed previously Received 25 November 1975.

(Castellanos et al., 1971b; Agha et al., 1972; Castellanos et al., 1972). After explaining the procedure and obtaining consent three tripolar catheter electrodes were introduced to obtain bipolar electrograms from the vicinity of the AV node in the septal low right atrium (LRA), 1 mm and/or 11 mm apart, as recorded by the His bundle electrographic lead (HBE); from the high right atrium (HRA) close to the superior vena cava, and from the low left atrium (LLA) explored through the coronary sinus in a position from which electrical stimulation produced negative P waves in leads I, II, and III (Agha et al., 1972). In 4 cases, 2 HBE leads (with interelectrode distances of 1 and 11 mm, respectively) were recorded at the same time (Fig. 1). Filtered 40-500 or 400-500 Hz intracardiac electrograms were obtained simultaneously, with at least three surface leads at a paper speed of 100 mm/s.

The moment of arrival of excitation at the recording electrodes (considered to have occurred at the

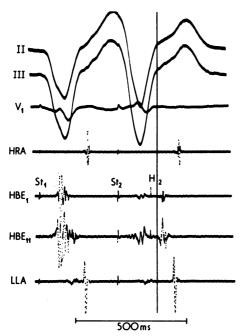


FIG. 1 Arrival of excitation at the recording atrial sites during retrograde (VA) conduction through the AV node-His Purkinje system. HRA=high right atrium; HBE₁ and HBE₁₁=electrograms 1 and 11 mm apart recorded by the His bundle electrographic leads. LLA=low left atrium. St₁=driving stimuli. St₂=testing stimulus. The onset of the corresponding atrial electrograms was as follows: (1) low septal right atrium in HBE₁₁; (2) low septal right atrium in HBE₁₁; (3) LLA; and (4) HRA. This was more evident after St₂ because the corresponding deflections were farther apart from the QRS complex.

onset of the corresponding electrograms) was studied during sinus rhythm as well as during atrial and ventricular pacing with the extrastimulus method in 12 patients with (presumably) chronic conducting disease who showed retrograde (VA) conduction during ventricular pacing. The latter was a prerequisite for this study. Retrograde activation of the atria was considered to have been present when the sinus sequence of arrival of excitation (HRA to septal LRA) changed to a septal LRA to HRA sequence. Spontaneous or induced runs of tachycardia were not seen in these patients.

Because not all retrograde His bundle and septal LRA deflections could be identified in driven beats, the time elapsing between inscription of the corresponding electrograms was measured following premature beats with coupling intervals short

enough to allow the retrograde H₂ deflections to emerge after the end of the ventricular electrograms (Castillo and Castellanos, 1970).

The following nomenclature will be used in this report:

A, H, and V represent the onset of septal LRA, His bundle, and ventricular deflections in the HBE during sinus rhythm.

HRA₁, HRA₂, septal LRA₁, septal LRA₂, LLA₁-LLA₂, H₁, and H₂ are the electrograms in the corresponding sites produced by driving (St₁) and testing (St₂) impulses. HBE₁ and HBE₁₁ refer to the interelectrode distances with which these leads were recorded.

Results

During sinus rhythm the duration of the AH intervals was normal according to the values presently used in our laboratory (60 to 120 ms). The sequence of arrival of excitation was from HRA to septal LRA to LLA. A similar sequence was observed when the HRA was paced.

Since this was a prerequisite in this study, retrograde (VA) conduction was seen in all patients during right ventricular apical pacing with the extrastimulus method. Retrograde AV nodal conduction time (given by the retrograde H₂-septal LRA₂ interval) was normal (between 70 and 110 ms, Table). The septal LRA₂ deflection was inscribed 25 to 55 ms (average 37.9 ms) before LLA₂ and 35 to 75 ms (average 53.7 ms) before HRA₂ (Table, Fig. 1 and 2). The LLA₂ electrogram preceded

TABLE Differences in arrival of excitation at recording sites during retrograde atrial activation (in ms)

Case No.	H_2 - LRA_2	LRA_2 - LLA_2	LRA_2 - HRA_2	LLA_2 - HRA_2
1	90	25	75	50
(atrial echo)	_	25	75	50
`2	85	35	55	20
(atrial echo)		35	55	0
3	85	55	55	0
(atrial echo)		55	55	0
4	110	30	60	30
5	95	25	55	25
6	105	30	40	10
7	110	35	35	0
8	105	40	70	30
9	70	50	70	20
10	95	40	40	0
11	90	50	40	-10
12	85	40	50	10
Average	93.7	37.9	53.7	15.4

HRA₂ in 8 patients, occurred at more or less the same time in 3 patients, and was inscribed 10 ms later in 1 case. The morphology of the P waves in the standard leads could not be determined with certainty since on most occasions they appeared on top of the T waves.

In all cases a decrease in the coupling (St₁-St₂) interval was associated with an increase of the septal LRA₁-LRA₂, LLA₁-LLA₂, and HRA₁-HRA₂ intervals which was of the same order of magnitude. As mentioned previously, retrograde activation of the His bundle was seen at the shorter coupling intervals in all patients but the H₁-H₂ intervals could not be measured because H₁ was frequently buried within the ventricular electrogram of the HBE lead.

Although runs of tachycardia were not elicited in the patients studied, single atrial echoes were induced by premature atrial stimulation in three cases (Table). When this occurred the sequence of arrival of excitation at the recording atrial sites was similar to the one shown by the same patients during ventricular pacing (Fig. 2 and 3).

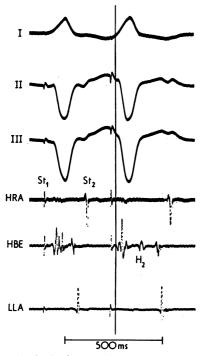


FIG. 2 Arrival of excitation at the recording sites during retrograde (VA) conduction following driving and premature stimuli.

Discussion

Recent studies have shown that some symptomatic patients with Wolff-Parkinson-White (WPW) syndrome might have unidirectional antegrade block in the accessory pathway, the latter being able to conduct exclusively in a retrograde direction (Slama, Coumel, and Bouvrain, 1973; Spurrell, Krikler, and Sowton, 1974a; Wellens and Durrer, 1974; Coumel and Attuel, 1974; Narula, 1974; Neuss, Schlepper, and Thormann, 1975; Svenson et al., 1975; Tonkin et al., 1975a, b).

Since intractable recurrent AV tachycardia can occur in these cases, surgical modes of therapy have been developed. Thus, identification of the accessory pathways which are functionally silent during antegrade conduction is of clinical importance. However, to accomplish this an understanding of the normal sequence of retrograde atrial activation is necessary.

The results of the present study show that in the absence of pre-excitation, activation of the septal LRA in the vicinity of the AV node (as recorded by the HBE lead) preceded that of the LLA (as recorded from the coronary sinus) and, of course, that of the HRA (Table, Fig. 1 and 2).

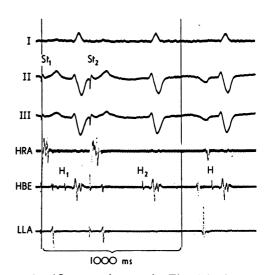


FIG. 3 (Same patient as in Fig. 2.) Arrival of excitation at the recording sites during driving (St_1) and testing (St_2) impulses delivered to the HRA and after an (atrial) echo induced by the second atrial impulse. The sequence of retrograde atrial activation of the echo was similar to that seen in the same patient during ventricular pacing (Fig. 2).

Moreover, in the 3 patients with atrial echo beats (elicited by premature atrial stimulation) the sequence of arrival of excitation at the recording electrodes was similar to that occurring in the same cases during the retrograde atrial activation of premature ventricular beats (Table and Fig. 2 and 3). Thus, it seems that the retrograde AV nodal-atrial pathway used by the returning impulse was the same in both situations. This is in keeping with the presence of a common 'upper pathway' located above the site where functional longitudinal dissociation of the AV node occurs (Scherf and Shookhoff, 1926; Schamroth, 1971). However, a larger number of patients should be studied to determine whether this is seen in all cases as well as in patients with accessory atrio-AV nodal or atrio-His bundle pathways.

It should be stressed that the differences in arrival of excitation at different sites are subjected to several variables that might explain the range of values shown in the Table. Foremost among these is the inevitability of some degree of catheter movement during the procedure (Agha et al., 1972). Even with great care it is possible, if the procedure is prolonged or the patient is not fully co-operative, for the electrode to shift its position from the beginning to the end of the procedure. In our department this happened more often with the HRA electrode (Agha et al., 1972). In retrospect, this factor might explain why in Case 11 (Table) the LLA2 electrogram was inscribed 10 ms after that of HRA2. Yet, the possibility of conduction through a preferential intra-atrial pathway (though less likely) cannot be completely excluded. A useful suggestion would be to compare radiographic films obtained at the beginning and at the end of the strip, which was not done in this study.

Furthermore, the coronary sinus electrode might not have been in an equivalent position in all of our cases. For instance, Tonkin et al. (1975b) and Svenson et al. (1975) have recorded several simultaneous electrograms from within the coronary sinus in patients with parietal left ventricular accessory pathways. They observed that the proximal coronary sinus to septal LRA intervals were shorter than distal coronary sinus to septal LRA intervals.

Another factor which has to be considered in evaluating differences shown in the Table is given by the method of recording electrograms as well as by the moment in which arrival of excitation at the electrodes as believed to have occurred. In some of our cases in which two HBE leads were recorded, the onset of retrograde bipolar atrial electrograms 1 mm apart occurred 15 to 20 ms after that of bipolar electrograms 11 mm apart, even when recorded simultaneously through the same catheter

(Fig. 1). These differences can also be noted during sinus rhythm since the inscription of the atrial electrograms 1 mm apart occurred after that of the electrograms 11 mm apart (Fig. 4).

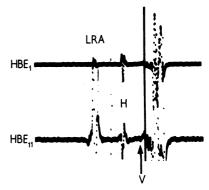


FIG. 4 Magnification of the onset of septal low right atrial (LRA) deflections in HBE_1 and HBE_{11} during sinus rhythm. Note that the atrial electrograms recorded by the former start with the rapid deflections of the electrogram recorded with electrodes 11 mm apart. Thus, the corresponding AH interval had a shorter duration.

That is, the AH intervals were shorter and the H_2 - A_2 intervals were longer when electrodes 1 mm apart were used. This might explain why Akhtar et al. (1975), who used electrodes 10 mm apart, recorded shorter H_2 - A_2 intervals (range 15 to 45; average 37.9 ms) than those observed in this study. However, the possibility of retrograde conduction through an antegradely blocked accessory atrio-His pathway could not be excluded in the cases of Akhtar et al. This raises the question as to whether patients with overt atrio-His pathways in whom both AV and VA conduction occurs via the bypass tract have a retrograde atrial activation pattern similar to that shown in Fig. 1 and 2.

In spite of this critical analysis, catheter recordings can be used to differentiate between the retrograde atrial activation pattern of impulses emerging from the AV node and that of those reaching the atria through a left sided (parietal) accessory pathway (WPW Type A). Yet, ectopic atrial impulses originating close to the area where the accessory pathway enters the left atrium could give a similar (in respect of catheter recordings) retrograde activation sequence. This can be seen in Fig. 83 of the monograph by Latour and Puech (1957) and in Fig. 6 of the article by Castellanos et al. (1971a).

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